

Reversible myocardial perfusion defect in a patient with anomalous origin of left circumflex coronary artery from right coronary sinus

Odwracalny ubytek perfuzji miokardium u pacjenta z nieprawidłowym odejściem gałęzi okalającej od prawej zatoki wieńcowej

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Abstract

We present the case of a 50 year-old woman with known anomalous origin of left circumflex coronary artery from the right sinus of Valsalva, with retro-aortic course confirmed by coronary computed tomography angiography. Due to progressive dyspnoea and a suspicion of ischaemia, the patient was referred to the Nuclear Medicine department for stress-rest myocardial perfusion scintigraphy. The imaging revealed inducible perfusion deficits mainly in the lateral and infero-lateral walls involving 14% of the total left ventricular (LV) myocardium. Although the detected coronary anomaly is considered nonmalignant, the extent of inducible ischaemia exceeding 10% of the LV myocardium should be taken into consideration while managing the patient.

Key words: anomalous coronary artery, inducible ischaemia

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Introduction

Coronary artery anomalies (CAAs) are a group of variable congenital disorders with diverse pathophysiological mechanisms and clinical manifestations [1]. The incidence of CAAs is approximately 0.3–0.9% in patients without structural abnormalities of the heart, but they are significantly more frequent (3–36%) in patients with structural heart disease. Although in young athletes CAAs are the second most common cause of sudden cardiac death (SCD), in middle aged or elderly individuals they are very rarely fatal. The type of coronary anomaly most commonly associated with SCD is the anomalous origin of a coronary artery (AOCA), especially one with a course between the aorta and the pulmonary artery.

Case report

We present the case of a 50 year-old female with no history of coronary artery disease (CAD), with known anomalous origin of left circumflex (LCx) coronary artery from the right sinus of Valsalva, with retro-aortic course confirmed by coronary computed tomography angiography (CCTA) (Figures 1, 2). The patient was referred to the Nuclear Medicine department for stress-rest myocardial perfusion scintigraphy to assess the degree of suspected ischaemia due to progressive dyspnoea. Treadmill stress test was not performed because of total hip replacement and post-surgery insufficiency. Single-photon emission computed tomography (SPECT) was performed in a 1-day protocol, with dipyridamole stress test (0.56 mg/kg of body weight) and under rest conditions, one hour after



Figure 1A, B. Coronary computed tomography angiography (CCTA): cross-section (A) and 3-dimensional reconstruction (B) showing anomalous origin of left circumflex (LCx) coronary artery from right sinus of Valsalva with retro-aortic course; RCA — right coronary artery

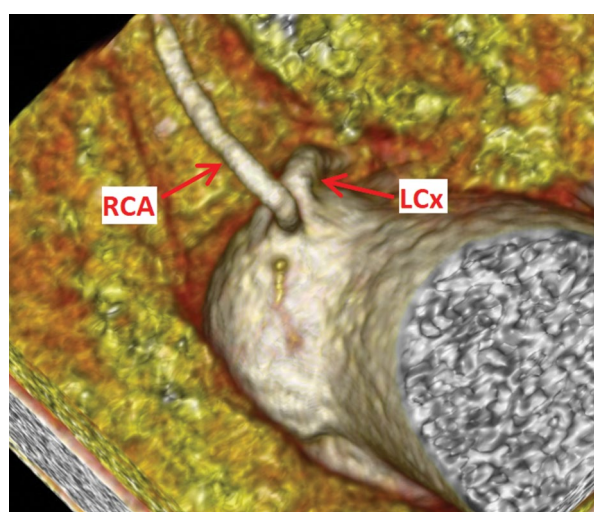


Figure 2. Coronary computed tomography angiography (CCTA): 3-dimensional colour reconstruction showing precisely the anomalous origin of left circumflex (LCx) coronary artery from right sinus of Valsalva next to normal origin of the right coronary artery (RCA)

injection of 333 and 999 MBq of ^{99m}Tc -MIBI (methoxy-isobutyl-isonitrile), respectively. Imaging revealed inducible perfusion deficits in two regions: the larger one in the lateral and infero-lateral walls, and the smaller one in the anterior and antero-lateral walls, involving 14% of the total left ventricular (LV) myocardium (Figure 3). The patient was submitted to medical treatment with valsartan, amlodipine, and bisoprolol.

Discussion

Reversible ischaemia corresponding to limited coronary flow reserve (CFR) in the lateral and infero-lateral walls of the LV, the region supplied by LCx, most probably resulted from the anomalous origin of this artery. Although the

detected coronary anomaly is generally considered non-malignant and haemodynamically insignificant, an extent of inducible ischaemia exceeding 10% of the LV myocardium is associated with a high risk of cardiac death and myocardial infarct (annual mortality > 3%) [2], which should be taken into consideration while managing the patient. The smaller reversible perfusion defect in the anterior (especially its apical segment) and the antero-lateral walls was most probably induced by myocardial bridging of the left anterior descending artery (LAD), also detected in CCTA.

It is worth pointing out that the patient was also diagnosed with mitochondrial myopathy with mutation of gene *FBN1* (manifesting with muscle weakness), which could potentially influence the biokinetics of regional and/or global MIBI uptake in cardiomyocyte mitochondria [3–5]. MIBI is a cationic compound capable of passive diffusion into the cytoplasm and mitochondria and is trapped inside the mitochondria due to negative plasma and mitochondrial membrane potentials. We did not observe disturbances in regional myocardial tracer uptake other than those described above. However, to quantitatively assess myocardial perfusion and to estimate the absolute value of myocardial blood flow (MBF) in mL/min/g in all LV segments, one should perform positron emission tomography (PET) [6]. When considering the appropriate perfusion tracer for cardiac PET scan in this case, one should avoid compounds which present with mitochondrial affinity, e.g. ^{18}F -flurpiridaz [binds to mitochondrial complex 1 (MC-1)], and choose between ligands with non-mitochondrial mechanisms of uptake, e.g. ^{82}Rb (potassium analogue) or ^{13}N ammonia (freely diffusing agent, which gets trapped inside the cell after conversion through glutamine synthase to ^{13}N -glutamine), or ^{15}O water (freely diffusing agent).

Conflict(s) of interest

The authors declare no conflict of interest.

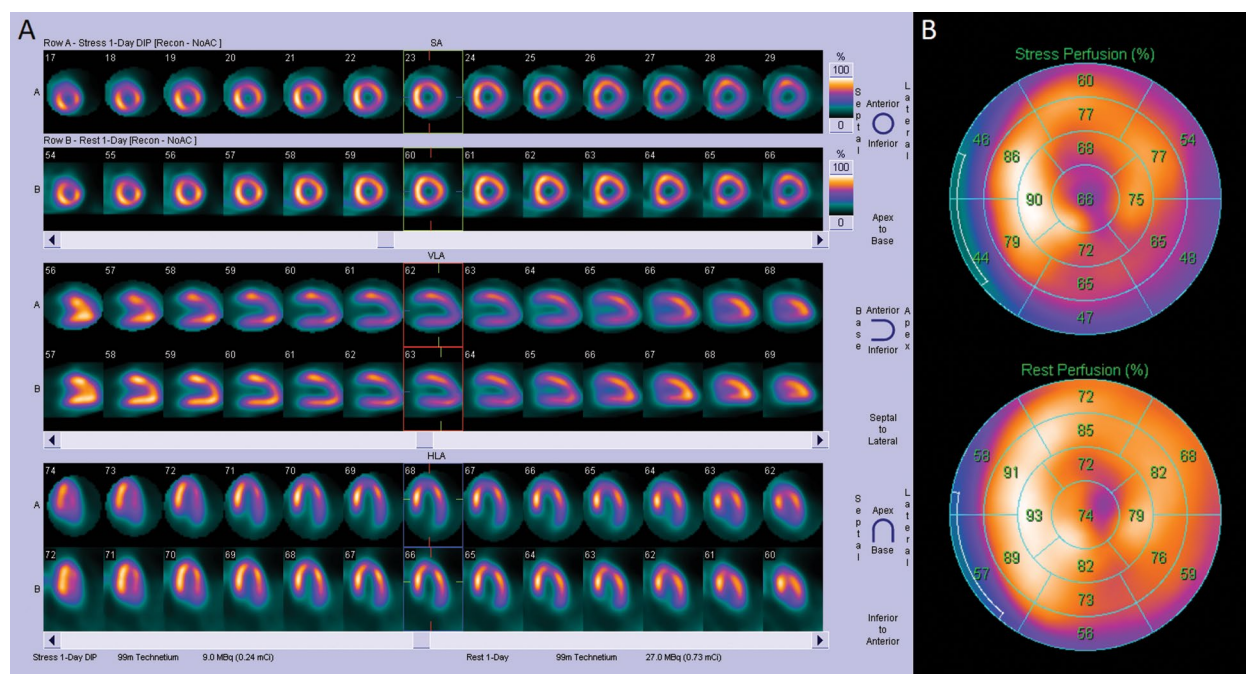


Figure 3A, B. Myocardial perfusion scintigraphy – single-photon emission computed tomography (SPECT): left ventricular (LV) slices (A), bull's eyes (polar maps; B). Inducible perfusion deficits in lateral and infero-lateral walls, and in anterior and antero-lateral walls, involving 14% of the total LV myocardium

Streszczenie

Prezentujemy przypadek 50-letniej kobiety z nieprawidłowym odejściem gałęzi okalającej od prawej zatoki wieńcowej z zaaoortalnym przebiegiem potwierdzonym w angiotomografii komputerowej tętnic wieńcowych. Z powodu postępującej duszności i podejrzenia niedokrwienia mięśnia sercowego pacjentkę skierowano do zakładu medycyny nuklearnej w celu wykonania wysiłkowo-spoczynkowej scyntygrafii perfuzyjnej serca. Badanie obrazowe uwidoczniało odwracalne zaburzenia perfuzji głównie w ścianach dolnej i dolno-bocznej obejmujące 14% mięśnia lewej komory serca. Mimo że wykryta anomalia tętnic wieńcowych jest uznawana za łagodną, to zakres odwracalnego niedokrwienia przekraczający 10% mięśnia lewej komory serca powinien być uwzględniony w trakcie postępowania z pacjentem.

Słowa kluczowe: anomalia tętnic wieńcowych, indukowane niedokrwienie

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References

1. Villa ADm, Sammut E, Nair A, et al. Coronary artery anomalies overview: The normal and the abnormal. *World J Radiol.* 2016; 8(6): 537–555, doi: [10.4329/wjr.v8.i6.537](https://doi.org/10.4329/wjr.v8.i6.537), indexed in Pubmed: [27358682](https://pubmed.ncbi.nlm.nih.gov/27358682/).
2. Montalescot G, Sechtem U, Achenbach S, et al. Task Force Members, ESC Committee for Practice Guidelines, Document Reviewers. 2013 ESC guidelines on the management of stable coronary artery disease: the Task Force on the management of stable coronary artery disease of the European Society of Cardiology. *Eur Heart J.* 2013; 34(38): 2949–3003, doi: [10.1093/eurheartj/ehd296](https://doi.org/10.1093/eurheartj/ehd296), indexed in Pubmed: [23996286](https://pubmed.ncbi.nlm.nih.gov/23996286/).
3. Pfeffer G, Chinnery PF. Diagnosis and treatment of mitochondrial myopathies. *Ann Med.* 2013; 45(1): 4–16, doi: [10.3109/07853890.2011.605389](https://doi.org/10.3109/07853890.2011.605389), indexed in Pubmed: [21867371](https://pubmed.ncbi.nlm.nih.gov/21867371/).
4. Ikawa M, Kawai Y, Arakawa K, et al. Evaluation of respiratory chain failure in mitochondrial cardiomyopathy by assessments of 99mTc-MIBI washout and 123I-BMIPP/99mTc-MIBI mismatch. *Mitochondrion.* 2007; 7(1–2): 164–170, doi: [10.1016/j.mito.2006.11.008](https://doi.org/10.1016/j.mito.2006.11.008), indexed in Pubmed: [17280875](https://pubmed.ncbi.nlm.nih.gov/17280875/).
5. Matsuo S, Nakajima K, Kinuya S, et al. Cardiac scintigraphic findings of mitochondrial myopathy, encephalopathy, lactic acidosis and stroke-like episodes: A case report. *Exp Clin Cardiol.* 2008; 13(2): 93–95, indexed in Pubmed: [19343124](https://pubmed.ncbi.nlm.nih.gov/19343124/).
6. Maddahi J, Packard RRS. Cardiac PET perfusion tracers: current status and future directions. *Semin Nucl Med.* 2014; 44(5): 333–343, doi: [10.1053/j.semnuclmed.2014.06.011](https://doi.org/10.1053/j.semnuclmed.2014.06.011), indexed in Pubmed: [25234078](https://pubmed.ncbi.nlm.nih.gov/25234078/).